



Case report

Apoplexia uteri: A rarely described post-mortem finding



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ABSTRACT

We present a case of apoplexia uteri, a rarely described condition of haemorrhagic necrosis in an atrophic endometrium and myometrium associated with terminal stress. This entity is well recognised in older literature but few recent publications have addressed this condition. It is thought to occur in association with hypoperfusion with passive hyperaemia and reperfusion injury.

This case serves to highlight this rarely encountered entity as a possible cause of haemorrhage in an atrophic endometrium in the 'perimortem' period. Incidental findings are occasionally observed in the course of forensic autopsy practice and knowledge of rarely encountered entities, such as that described in this case, is essential to prevent diagnostic uncertainty and misdiagnosis.

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1. Introduction

We describe a case of apoplexia uteri in an atrophic endometrium discovered at post-mortem. Apoplexia uteri is a rarely encountered condition which is most commonly described in the older literature.^{1,2} Few recent publications are available on the subject. Cases to date describe haemorrhagic necrosis of the post-menopausal atrophic endometrium.^{1–3} It is reported as occurring in association with terminal stress. The underlying mechanism is postulated to involve hypoperfusion with passive hyperaemia and reperfusion injury.

We report a case of a 33-year-old female who underwent a coroner's autopsy. At autopsy there was evidence of cirrhosis and hepatorenal syndrome and also evidence of cerebral hypoxia. The endometrium and myometrium showed changes of apoplexia uteri.

'post-mortem' there was evidence of cirrhosis and hypoxic brain injury. In addition two foci of endometrial and myometrial haemorrhage were identified (see Fig. 1). These two foci of haemorrhage on the anterior and posterior walls of the uterus were symmetrical in shape. This symmetry is somewhat unusual and suggested an associated traumatic or hormonal injury from an intrauterine contraceptive device; however such a device was not seen at the time of post-mortem. Histological sections from this area of haemorrhage demonstrated a discrete focus of inner layer myometrial infarction with extensive haemorrhage and extravasation of red blood cells into the affected endometrium and adjacent myometrium (Figs. 2–4). The infarct seen was not associated with an inflammatory response suggesting that it was a terminal event. The adjacent non-haemorrhagic endometrium showed changes consistent with atrophy despite her age. There was no evidence of a primary vasculitic process or a vascular lesion.

2. Case report

The case was that of a 33-year-old woman who died following an episode of acute cerebral hypoxia. Her clinical history was significant for hepatorenal syndrome and alcohol consumption. At

3. Discussion

The microscopic features seen in the inner myometrium and endometrium are most in keeping with apoplexia uteri, a condition of postmenopausal bleeding in an atrophic endometrium related to hypoperfusion.³ Although predominantly an endometrial lesion, literature to date also describes variable extension to involve adjacent myometrium,^{1–3} as seen in the case presented. Apoplexia

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Fig. 1. Transverse section through the uterine cavity. A discrete focus of haemorrhage is seen involving both the endometrium and myometrium on the anterior and posterior uterine walls.

uteri, or haemorrhagic necrosis of the endometrium, developing in the absence of a vascular occlusion, has been well recognised at autopsy in older patients and is documented particularly in older (mostly German) literature.³ In spite of this, little recent literature is available on the subject.

The most recent publication addressing this entity was in 2004. An autopsy study by Peych et al. from Kolin in the Czech Republic³ assessed 47 cases of macroscopically visible intra-endometrial haemorrhage in atrophic uterine cavities. In their cohort 61% of the deceased patients had suffered various cardiovascular diseases or acute abdomen. There were also associated vascular changes in other organs in 46% of cases, mainly affecting the gastrointestinal tract. All 47 cases showed endometrial and myometrial congestion histologically. In 38 cases this was accompanied by haemorrhage into the endometrial stroma with occasional extension into the myometrium. In some instances the histological picture resembled that of a haemorrhagic infarction.

The postulation of that study was that this haemorrhage into the endometrium and myometrium in an atrophic uterus was as a

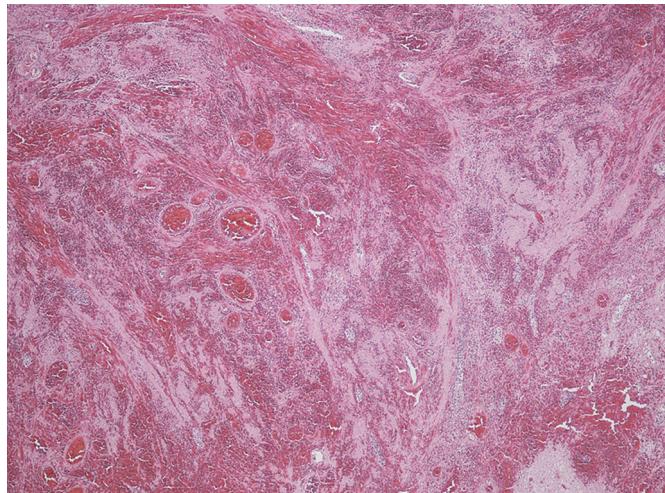


Fig. 3. Haematoxylin and eosin stained image of the inner myometrium showing extensive haemorrhage. Admixed vascular channels are dilated and congested. Magnification $\times 200$.

result of hypoperfusion leading to passive hyperaemia and reperfusion injury.

An older autopsy study carried out in 1968 by Daly et al.¹ also examined autopsy cases. In this instance the uteri of 379 patients over the age of 50 years were examined. Of these, 19 (5%) were found to demonstrate endometrial haemorrhage with or without necrosis.

Again the histological changes presented in this paper describe extravasation of erythrocytes, with associated leucocyte infiltration in many cases and inconstant necrosis focally in the endometrium. There was variable involvement of only superficial portions or full thickness of endometrium with myometrial involvement in some cases.

Interestingly in this study they also describe cases with a clinical history of cardiovascular decompensation in the period preceding death. Of note six cases also had evidence of haemorrhagic necrosis of the bowel wall in addition to the endometrial haemorrhage.

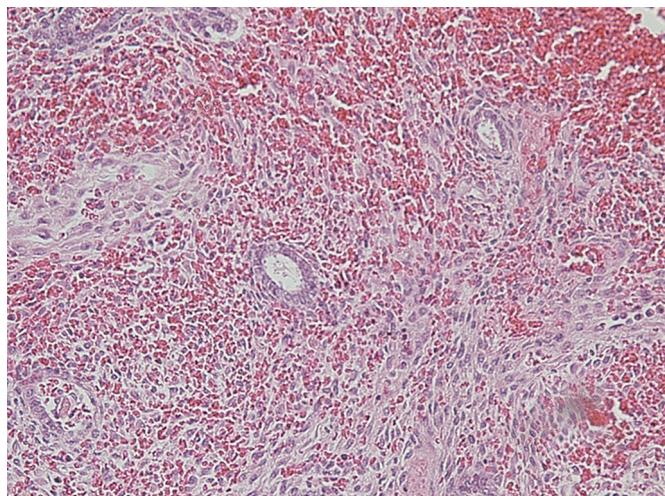


Fig. 4. Haematoxylin and eosin stained image of atrophic type endometrium showing haemorrhage characterised by red blood cell extravasation within the interstitium. Vascular channels seen show no evidence of vasculitis or occlusion. Magnification $\times 400$.

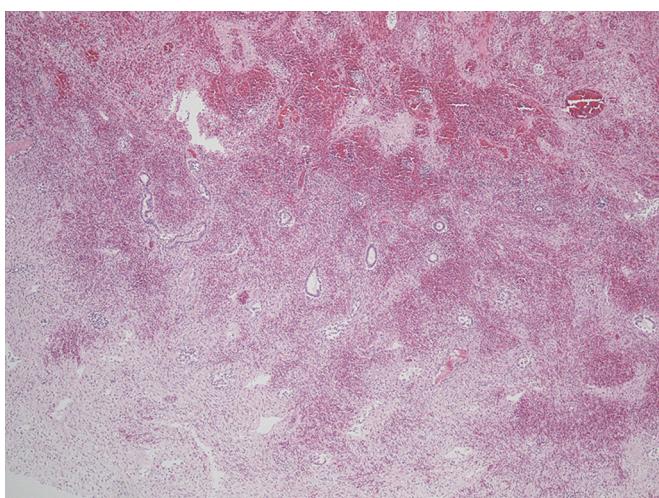


Fig. 2. Haematoxylin and eosin stained microscopic image demonstrating interstitial haemorrhage with extravasation of red blood cells involving the junction of endometrium and the inner layer of myometrium. Magnification $\times 200$.

Postmenopausal uterine bleeding related to atrophic endometrium is also well documented clinically.^{6–9} This was described by Choo et al. in 1985⁴ where they report 82% of cases of postmenopausal uterine bleeding examined demonstrating atrophic endometrium as the cause.

Meyer et al. in 1971⁵ similarly studied 37 patients with unexplained postmenopausal bleeding and compared these with 40 control patients with atrophic endometrium. Meyer found that there was a significant increased incidence in diabetes and hypertension in the bleeding cohort. He also demonstrated evidence of endometrial vascular disease on histological sections in 21% of these patients. Other possible aetiologies put forward in this study included uterine prolapse leading to passive congestion and rupture of endometrial cysts.

In the case we present, histological changes of apoplexia uteri are evident. The aforementioned autopsy series from the literature refer to cardiac disease or decompensation in all of the cases they describe and the underlying mechanism involved is postulated as being related to a generalised hypovolaemic state. In the case we present there was a 'pre-mortem' history of cirrhosis and hepatorenal syndrome with superimposed acute cerebral hypoxia at the time of death. This presents a probable explanation for the postulated hypoperfusion associated with this condition. The underlying mechanisms thought to be associated with clinically described cases of haemorrhage in atrophic endometrium, such as uterine prolapse and endometrial cysts, were not recognised at post-mortem in this case. No intrauterine contraceptive device was seen at post-mortem to account for the symmetry of the lesion seen. It is interesting to note that no evidence of intestinal ischaemia was found in the case presented, an accompanying finding that is well described. However, multiple petechial haemorrhages were identified within the mesenteric adipose tissue.

4. Conclusion

We present a case of apoplexia uteri, a rarely described condition involving haemorrhagic necrosis of the postmenopausal atrophic endometrium and myometrium, reported to occur with

terminal stress. The underlying mechanism is postulated to involve hypoperfusion and passive hyperaemia and reperfusion injury.

This entity is well recognised in older literature but few recent publications have addressed this condition. Incidental findings are occasionally found at the time of autopsy in medicolegal practice that may not be related to the cause of death, but are, however, important to recognise. As apoplexia uteri is a condition recognised to occur in association with terminal stress it is vital to be aware of this condition as a possible cause of haemorrhage in an atrophic endometrium in the perimortem period in patients with underlying hypoperfusion.

Ethical approval

Not required.

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Conflict of interest

The authors have no financial or personal relationships with other people or organisations that could inappropriately influence this work.

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